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SECOND AUTUMN WORKSHOP ON MATHEMATICAL ECOLOGY

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"Regulation and Stability of a Free-Living Host-Parasite System: *Trichostrongylus tenuis* in Red Grouse II. Population Models"

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Regulation and stability of a free-living host–parasite system: *Trichostrongylus tenuis* in red grouse. II. Population models

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Summary

1. The population dynamics of red grouse, *Lagopus lagopus scoticus* and the parasitic nematode, *Trichostrongylus tenuis* were explored to determine whether interactions between the parasite and host were sufficient to generate cycles in grouse abundance. Two alternative models were used that explicitly consider the dynamics of either the free-living, or arrested larval stages of the parasite.

2. Providing that the life expectancy of the free-living larvae is more than 2–4 weeks, the parasite can readily establish in grouse populations. Larval arrestment tends to reduce the intrinsic growth rate of the parasite and thus increases the size of the host population required for the parasite to establish.

3. Grouse numbers will tend to cycle when the parasites exhibit low degrees of aggregation and parasite-induced reductions in host fecundity are greater than parasite-induced increases in host mortality. The population cycles produced in the model have the slow increase followed by a rapid decline characteristic of the grouse population studied at Gunnerside.

4. The period of the cycles is determined by the intrinsic growth rate of the grouse population and either larval life expectancy (Model I), or the duration of larval arrestment (Model II). Cycle periods decrease as host population growth rate increases, and lengthen with increases in either free-living larval life expectancy, or the duration of larval arrestment. If the duration of larval arrestment is sufficiently long (>6 months), the cycles die out and the dynamics of the grouse–nematode system are very stable.

5. Estimates of all of the model's parameters may be made from long-term records of grouse populations. Numerical analysis of the model's behaviour suggest that a model with limited arrested larval stages more closely corresponds to the grouse populations in the North of England. The 4–5 year cycles exhibited by these populations will be more sporadic, or absent on estates where the parasite is unable to establish.

6. The analysis shows that the empirical data collected on *T. tenuis* are consistent with it being the cause of the cycles observed in grouse populations in the North of England.

Key-words: grouse, population cycles, parasites, helminths.

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Introduction

Controversy exists over whether the long-term cycles of abundance observed in grouse populations are caused by extrinsic interactions between grouse and their nematode parasite (Hudson, Dobson & Newborn 1985), or by intrinsic features of grouse population biology (Wynne-Edwards 1986; Watson & Moss 1979; Watson, Moss, Rothery & Parr 1984). The population biology of red grouse and its caecal nematode *Trichostrongylus tenuis* have been studied in detail at Gunnerside, North Yorkshire for 10 years. Losses from the grouse population were associated with the intensity of parasite infection,

while manipulation experiments have demonstrated that the parasite is the principal cause of reductions in grouse breeding production (Hudson, Newborn & Dobson 1992). In this paper we use the data presented in the Gunnerside study to develop a mathematical model that explores the population dynamics of the grouse–*T. tenuis* system. The main objective of this exercise is to determine whether interactions between *T. tenuis* and grouse are sufficient to generate the observed cycles in grouse abundance, and to examine which features of the parasite–host interaction are necessary for sustained cycles in parasite and host abundance.

Anderson & May (1978) and May & Anderson (1978) (henceforth A&M) derived the fundamental model for the population dynamics of parasitic helminths, P, and their hosts, H. Here we extend the A&M models to consider the dynamics of the free-living, W, and arrested larval stages, A, of the parasite. In particular, we are concerned with the dynamic consequences of *hypobiosis*: the ability of larval parasites to arrest their development after infecting a suitable host (Schad 1977; Gibbs 1986). This period of arrestment has the potential to put significant time delays into the parasite life cycle and may cause time-delayed density-dependent effects to appear in the dynamics of the parasite–host interaction. The various birth and death rates of the grouse and *T. tenuis* populations and the flow rates between the different stages of the parasite life cycle are illustrated in Fig. 1 and Table 1.

Initially, the effects of larval arrestment are ignored and a modified three equation A&M model is described that considers the free-living larval stages of the parasite (Model I). We then assume the free-living larvae are relatively short-lived and describe a second three equation model in which the larvae enter a period of hypobiosis immediately after infecting a suitable host (Model II). The population dynamic consequences of a proportion of the larvae entering hypobiosis are examined in the final section of the paper. Seasonal variation in the survival of the free-living stages and in the propensity of larvae to arrest their development will be considered in a subsequent publication (Dobson & Hudson, unpublished).

STRUCTURE OF THE BASIC MODEL

Model I. Long-lived free-living larvae

The dynamics of a host–parasite interaction that includes free-living infective larvae may be described by the following set of three coupled differential equations:

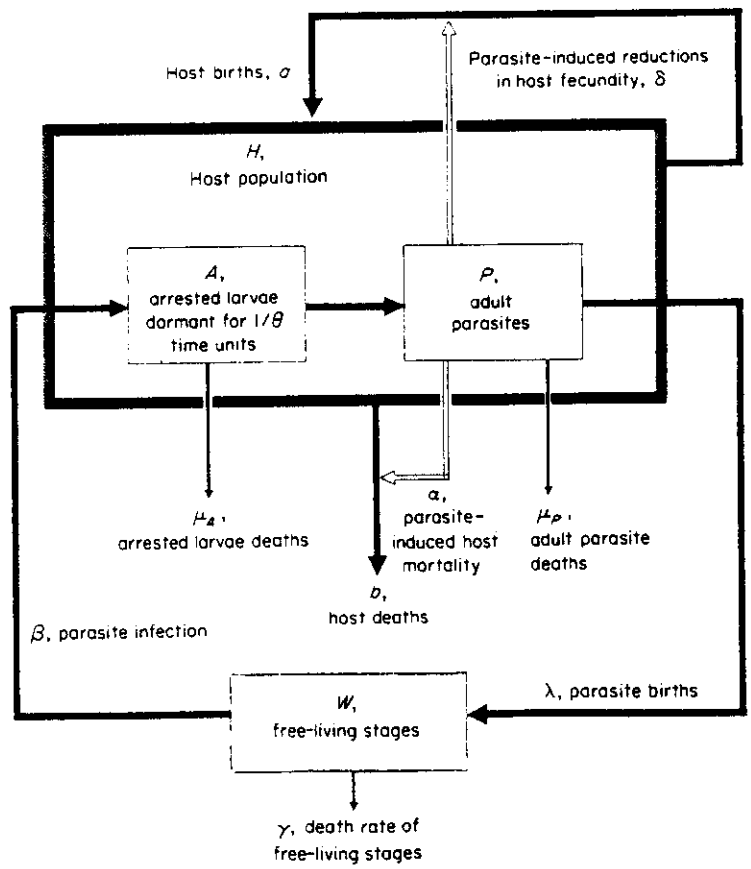

Fig. 1. Schematic representation of the life cycle of *T. tenuis* in red grouse illustrating the different birth, death and transmission rates occurring in the life cycle and the notation used to denote each process. Note that parasites can affect both host survival and breeding production.

Table 1. Notation used to denote various population parameters

Parameter	Description
H	Size of host population (numbers of grouse).
W	Numbers of free-living parasite stages (eggs & larvae).
A	Total numbers of arrested larvae.
P	Total numbers of adult worms.
a	Instantaneous birth rate of grouse (/unit of time).
b	Instantaneous death rate of grouse due to all causes except the parasite (/unit of time).
α	Instantaneous death rate of host due to the parasite (/worm/unit of time).
δ	Instantaneous reduction in grouse fecundity due to the parasite (/worm/unit of time).
Δ	Density-dependent reduction in grouse fecundity and survival (/host/unit of time).
λ	Instantaneous birth rate of parasite eggs (/unit of time).
μ_i	Instantaneous death rate of adult, μ_P, and arrested, μ_A, parasite stages in grouse (/unit of time).
γ	Instantaneous death rate of the free-living egg and larval stages (/unit of time).
β	Instantaneous rate of ingestion of parasite infective stages (/host/unit of time).
k	Parameter of the negative binomial distribution which measures inversely the degree of aggregation of parasites within the host population.
Θ	The rate at which arrested larvae develop into adult worms. (/unit of time).
σ	The proportion of larvae that arrest (Model III only).

$$dH/dt = (a-b)H - (\alpha+\delta)P \qquad \text{eqn 1}$$

$$dW/dt = \lambda P - \gamma W - \beta WH \qquad \text{eqn 2}$$

$$dP/dt = \beta WH - (\mu_P + b + \alpha)P - \alpha\frac{P^2}{H}\left(\frac{k+1}{k}\right) \qquad \text{eqn 3}$$

The parameters used to describe the different processes operating are defined in Table 1. The model assumes that the parasites are distributed in an aggregated manner, best described by the negative binomial distribution. This corresponds to the situation observed in the field data for *T. tenuis* (Hudson, Newborn & Dobson 1992). The degree of aggregation is given by k, a parameter which varies inversely with the degree of aggregation. Field experiments have shown that parasites reduce host breeding production and survival (Hudson 1986a,b; Hudson, Newborn & Dobson 1992); the available evidence suggests that these effects act linearly over two orders of magnitude, as is assumed in the standard A&M model. We have tested for non-linearity in this relationship by trying to fit a more complex function to the data. There is no indication that the addition of a non-linear term in the regression of birth rate on worm burden provides a significantly better description of the relationship.

Model II. The effects of hypobiosis

In model II we consider a second modification of the basic A&M framework containing an equation for the larvae that enter a period of arrestment in their development *after* infecting the definitive host and before developing to pathogenic adults. As arrested larvae are present within the host, they are subject to mortalities due to both the natural mortality rate of the hosts and parasite-induced mortalities due to the presence of adult parasites (e.g. when hosts die, the arrested larvae will also die). As metabolic activity in arrested larvae is very low they have no discernible effect on host survival or fecundity and their intrinsic mortality rate is extremely low (thus $\mu_P \gg \mu_A$). In this model the equation for host population size remains unaltered and equations 2 and 3 are replaced by

$$dA/dt = \frac{\lambda PH}{(H+H_0)} - (\mu_A + b + \theta)A - \alpha\frac{PA}{H} \qquad \text{eqn 4}$$

$$dP/dt = \theta A - (\mu_P + b + \alpha)P - \alpha\frac{P^2}{H}\left(\frac{k+1}{k}\right) \qquad \text{eqn 5}$$

This model assumes that the free-living stages are short-lived relative to other stages in the life cycle and transmission is governed by the single parameter H_0 ($=\gamma/\beta$). Immediately after establishing in the host the larvae enter a period of hypobiosis and remain arrested for a period of $1/\Theta$ years. Although it is possible that only a proportion of larvae enter hypobiosis, we initially choose to model the phenomenon as an 'all or nothing' effect.

Basic reproductive rate of the parasite, R_0

Before considering the model's transient and equilibrium dynamics, it is instructive to derive expressions for the basic reproductive rate of the parasite, R_0, and the threshold number of hosts, H_T, required to sustain a continuous parasite infection. Maximum rates of parasite growth occur when the parasite is first introduced into the population; at this time pathogenicity and other density-dependent constraints are effectively trivial. Collapsing equations 1–3 and 4–5 down under these conditions produces two expressions for the initial growth rate of the parasite population in models I and II, respectively

$$R_0 = \frac{\beta\lambda H}{(\mu_P + b + \alpha)(\gamma + \beta H)} = \frac{T_1}{M_1 M_2} \qquad \text{eqn 6}$$

$$R_0 = \frac{\theta\beta\lambda H}{(\mu_P + b + \alpha)(\gamma + \beta H)(\mu_A + b + \theta)} = \frac{\theta T_1}{M1 M_2 M_3} \quad \text{eqn 7}$$

The two expressions are similar in form: they consist of a 'birth' term, $T_1 = \beta\lambda H$, the rate of transmission of eggs from birth through to establishment in another grouse, and the mortality rates of each stage in the life cycle: adult worms, $M_1 = (\mu_P + b + \alpha)$, free-living larvae, $M_2 = (\gamma + \beta H)$, and arrested larvae, $M_3 = (\mu_A + b + \Theta)$. In both cases the basic reproductive rate of the parasite consists of the product of the mean number of new infections established by each female worm and the life expectancies of each stage of the worms life cycle. Estimates of some of these parameters are derived in Hudson, Newborn & Dobson (1992) or have been obtained from previously published literature (Table 2).

As is usually the case with epidemiological studies, few data are available with which to estimate rates of transmission. A coarse estimate of the rate of transmission may be obtained by determining the threshold host density, H_T at which the parasite is just able to maintain itself in the host population. Expressions for H_T in parasites with, or without, larval arrestment are obtained by setting R_0 to unity and rearranging equations 6 and 7.

$$H_T = \frac{\gamma M_1}{\beta(\lambda - M_1)} \quad \text{Model I, eqn 8}$$

$$H_T = \frac{\gamma M_1 M_3}{\beta(\lambda\theta - M_1 M_3)} \quad \text{Model II, eqn 9}$$

The threshold density of grouse at which infection rate is zero will vary between populations according to a variety of environmental conditions (Hudson, Dobson & Newborn 1985; Shaw, Moss & Pike 1989; Hudson, Newborn & Dobson 1992). Data collected from the Gunnerside population study suggest that the parasite infection rate is negligible at densities less than 24 grouse km^{-2}. This suggests a figure for β in the order of 0·1 ($0{\cdot}6-1{\cdot}6 \times 10^{-1}$).

As with most helminth parasites, the survival of the free-living larval stages is dependent upon temperature and humidity (Hudson 1986a; Shaw, Pike & Moss 1989). Field studies conducted in the north-east of Scotland by Shaw, Moss & Pike (1989) suggest that free-living larvae live for less than a month. In contrast, the laboratory experiments of Watson (1988) suggest the free-living larvae have a life expectancy of 2–6 months, while data from other Trichostrongyle species suggest that cool, moist conditions may allow free-living larvae to live for longer than this (Boag & Thomas 1985). The effect of variations in the life expectancy of the free-living stages on the ability of the parasite to establish may be examined by plotting the values of R_0 and H_T for life cycles with and without arrestment (Fig. 2). In both cases, once average larval life expectancy falls below 2–3 weeks the basic reproductive rate falls below unity and the parasite will not be sustained within the grouse population.

An interesting point to emerge from Fig. 2 is that parasite populations that exhibit hypobiosis have reduced growth rates when compared to populations with more direct life cycles. Examination of equation 7 suggests that low levels of host pathogenicity and high survival rates may be important prerequisites

Table 2. Population parameters for *T. tenuis* and red grouse

Parameter	Symbol	Estimated value (range)
Grouse fecundity	a	1·8 (0–2) $year^{-1}$
Grouse mortality	b	1·05 $year^{-1}$
Parasite fecundity	λ	11 (9·2–11·5) $year^{-1}$
Adult worm mortality	μ_P	1·0 (0·8–1·2) year
Arrested larvae mortality	μ_A	0·5 $year^{-1}$
Mortality of free-living parasite stages	γ	6·5–13 $year^{-1}$
Duration of arrestment	$1/\Theta$	2–6 months
Parasite pathogenicity	α	3×10^{-4} $worm^{-1}$ $year^{-1}$
Parasite reduction in host fecundity	δ	5×10^{-4} $worm^{-1}$ $year^{-1}$
Aggregation of parasites in hosts	k	1·0 (0·5–1·8)
Transmission rate	β	0·1 ($0{\cdot}6-1{\cdot}6 \times 10^{-1}$) $host^{-1}$ $year^{-1}$

The parameter estimates were obtained from the following sources: *a*, Hudson 1986a,b, Hudson, Newborn & Dobson 1992; *b*, Jenkins, Watson & Miller 1963, Hudson 1986a,b; λ, Hudson 1986b, Shaw, Moss & Pike 1989, Hudson & Dobson, in press; μ_P, Hudson, Newborn & Dobson 1992, Shaw, Moss & Pike 1989; μ_A, arrested nematode larvae have very low mortality rates (Gibbs 1986), by setting this parameter at 0·5 we assume this source of mortality is negligible; γ, Watson 1988, Shaw, Moss & Pike 1989; $1/\Theta$, Shaw, Moss & Pike 1989; α, δ & k, Hudson 1986a,b, Hudson, Newborn & Dobson 1992; β, this study. The estimates of parasite-induced mortality and reductions in host fecundity are obtained from the slopes of the relationships between parasite burden and host mortality and fecundity given in Hudson, Newborn & Dobson (1992). The estimates of host mortality and fecundity are obtained from extrapolating these relationships to the point where parasite burden is zero.

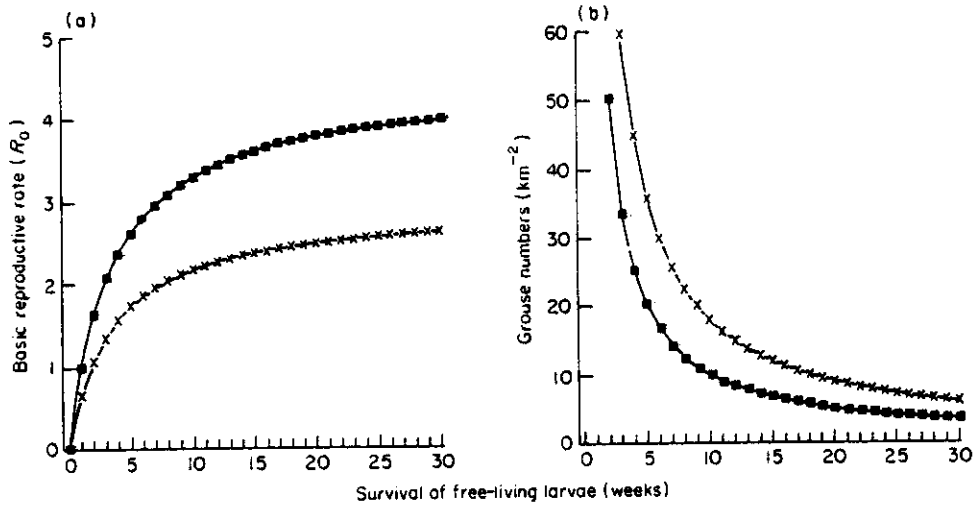

Fig. 2. The effect of changes in the life expectancy of the free-living larvae on (a) the basic reproductive rate of the parasite, and (b) the threshold host population required for parasites to establish for Model I (no arrestment) (■) and Model II (arrested larvae) (x). Parameter values are $H = 100$, $b = 1{\cdot}05$, $\alpha = 0{\cdot}0003$, $\lambda = 10$, $\beta = 0{\cdot}1$, $\mu_A = 0{\cdot}5$, $\mu_P = 1{\cdot}2$, $\Theta = 3$.

for the evolution of arrested development. The costs of hypobiosis may be further appreciated by considering the effect of duration of larval arrestment on R_0 (Fig. 3). The observed reduction in R_0 only reflects the additional mortality experienced by hypobiotic larvae; as discussed further below, the potential growth rate of the parasite population will be further reduced by the time delays inherent in larval arrestment.

Equilibrium densities of hosts and parasites

A qualitative comparison of how larval demography affects the magnitude of host and adult parasite populations may be gained by considering the equilibrium properties of both sets of equations. By setting $dH/dt = dW/dt = dP/dt = 0$ in equations 1–3, we obtain for model I:

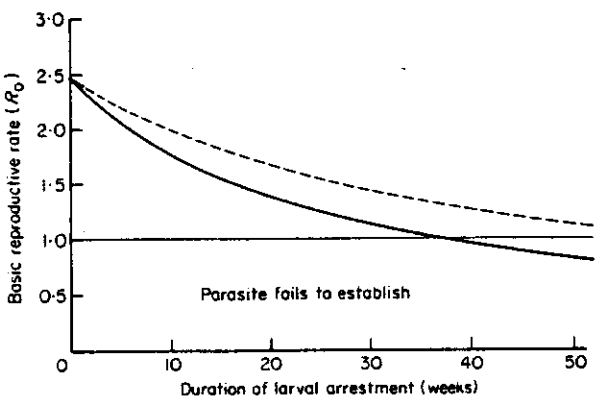

Fig. 3. The effect of duration of larval arrestment on the basic reproductive rate of *T. tenuis*. All parameters are as in Fig. 2 except for grouse density, H, which is set at 25, Θ is now a variable. The figure is drawn for two values of arrested larval mortality, $\mu_A = 0{\cdot}2$ (– – –) and $\mu_A = 1{\cdot}0$ (——), the actual mortality rate is very low and probably lies between these two values.

$$\frac{P^*}{H^*} = M_P^* = \frac{(a-b)}{(\alpha+\delta)} \qquad \text{eqn 10}$$

$$W^* = \frac{d'(a-b)}{\beta(\alpha+\delta)} \qquad \text{eqn 11}$$

$$H^* = \frac{\gamma d'}{\beta(\lambda - d')} \qquad \text{eqn 12}$$

Here M_P^* is the mean parasite burden of adult grouse at equilibrium and $d' = \mu_P + \alpha + b + k'(a-b)\alpha/(\alpha+\delta)$, with $k' = (k+1)/k$. In model II, where larvae enter a period of hypobiosis, an identical expression is obtained for mean burden of adult parasites, but modified expressions are obtained for equilibrium host density and the mean burden of arrested parasite larvae:

$$H^* = \frac{\gamma d' d''}{\beta(\lambda\theta - d'd'')} \qquad \text{eqn 13}$$

$$M_A^* = \frac{A^*}{H^*} = \frac{d'(a-b)}{\theta(\alpha+\delta)} \qquad \text{eqn 14}$$

Here d' is defined as above, while $d'' = \mu_A + b + \Theta + (a-b)\alpha/(\alpha+\delta)$. In both cases parasite burdens vary inversely with rates of parasite-induced reductions in host survival and fecundity. The numbers of free-living larvae varies directly with mean parasite burden and inversely with their rate of acquisition by the hosts. The expressions for equilibrium host population density closely resembles the expressions for the threshold for establishment (equations 8 & 9; Fig. 2). In both cases, H^* varies inversely with parasite fecundity, λ, and the rate at which parasite larvae are ingested, β.

Equilibrium host density is higher when the larvae enter hypobiosis; the size of the equilibrium host population increasing with the duration of arrestment (Fig. 4). This increase occurs as hosts live

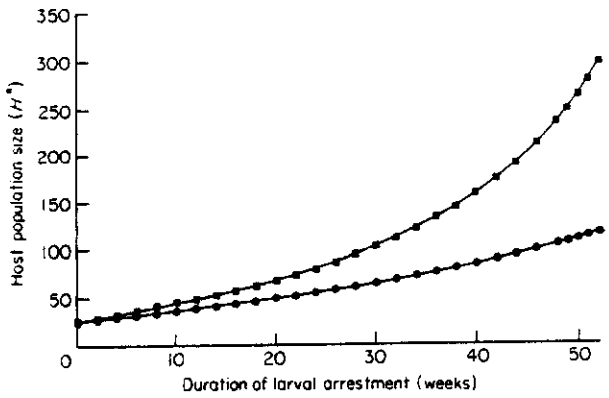

Fig. 4. The effect of duration of larval arrestment on the size of the host population at equilibrium (parameter values as in Fig. 2 with $a = 1{\cdot}4$ and $k = 1$). The figure is again drawn for two values of arrested larval mortality: $\mu_A = 0{\cdot}2$ (•) and $\mu_A = 1{\cdot}0$ (■). This figure assumes the parasite has no effect on host fecundity.

longer if the infective larvae enter an obligatory period of non-pathogenic arrestment before developing to pathogenic adults. Increased host life expectancy will roughly correspond to the period of larval arrestment.

Dynamic properties of the model

The dynamic properties of the model may be explored by both numerical simulation and local stability analysis of the equilibrium (the details of the latter are given in Appendix 1). Initially, we consider the effects of larval biology on the dynamics of a basic grouse–nematode system in the absence of any parasite-induced effects on host fecundity. Numerical simulations of the model were performed using the Numerical Methods package of MathSoft (1990). In each case an integration interval corresponding approximately to 1 week of real time was used. The simplest examples show two important features of the host–parasite interaction: (i) neither parasite-induced host mortality, nor arrested larval development lead to sustained oscillations in the abundance of either the hosts or the parasites (Fig. 5); (ii) although larval arrestment increases the propensity for cycles in parasite and host abundance, the period of these cycles is longer than those produced in the model without larval arrestment.

PARASITE-INDUCED REDUCTIONS IN HOST FECUNDITY

Experimental manipulations of parasite burdens in grouse have demonstrated that *T. tenuis* reduces the breeding production of infected female grouse (Hudson 1986a,b; Hudson, Newborn & Dobson 1992). When the effects of parasite-induced reductions in host fecundity are included in Models I

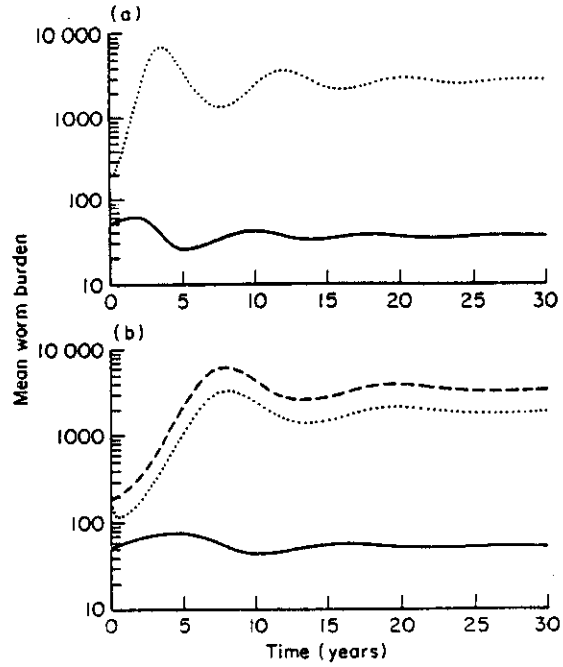

Fig. 5. Comparison of the dynamics of (a) Model I, and (b) Model II in the absence of any effect of the parasite on host fecundity. In both cases the host population is the lowest line, mean adult parasite burden is the dotted line, and mean burden of arrested larvae and adult worms is the dashed line in (b).

and II, both hosts and parasites show diverging cycles of abundance that drive both populations to extinction. More formally, it can be shown that in both models the numbers of hosts and parasites will cycle when the following inequality is transgressed (Appendix 1):

$$\alpha/\delta > k \qquad \text{eqn 15}$$

The cycles are thus dependent on the relationship between the parasites' effects on host survival and fecundity, and the degree of parasite aggregation within the host population. The destabilizing effects of parasite-induced reductions in grouse breeding production can only be offset by either high levels of parasite-induced host mortality, or high levels of parasite aggregation. Neither of these are characteristic of the grouse–*T. tenuis* system.

GROUSE TERRITORIAL BEHAVIOUR

Inclusion of the territorial behaviour of the grouse will tend to constrain the size of the host population, and may reduce the propensity for unstable cycles. This intrinsic form of host population regulation may be incorporated into the model by inserting an additional term into equation 1 which reduces grouse fecundity rates as density rises.

$$dH/dt = (a - b - \Delta H)H - (\alpha + \delta)P \qquad \text{eqn 16}$$

Inclusion of the density-dependent term mimics a fall in the availability of suitable territories to a point

where no further individuals can be recruited to the population (Davies 1978; Paterson 1980). In the absence of the parasite, the grouse will stabilize at carrying capacity K, where

$$K = H^* = (a - b)/\Delta \qquad \text{eqn 17}$$

Inclusion of the territorial behaviour of the grouse hosts allows a comparison of the effects of parasite-induced reductions in host fecundity and larval arrestment (Fig. 6). Both models show damped cycles in host and parasite abundance in the absence of parasite-induced effects on host fecundity. When this is included in the models, both exhibit either weakly damped or diverging cycles of host and parasite abundance. In contrast to the model without host regulation, parasite arrestment now acts to stabilize the system, giving rise to cycles of longer period that tend to fade out more rapidly.

The influence of larval biology on the dynamics of intrinsically regulated grouse populations is more subtle. In Model I, stability requires

$$\gamma > d'k\left(\frac{\alpha + \delta}{\alpha - \delta k}\right) \qquad \text{eqn 18}$$

This condition suggests that short larval life expectancy will tend to stabilize the system provided the condition in equation 15 is satisfied. As larval life expectancy increases, cyclic oscillations of host and parasite are likely to become more pronounced.

Model II requires slightly more complex criterion for stability, the simplest condition is that

$$\frac{\alpha}{\delta} > k\left(\frac{d' + d''}{d''}\right) \qquad \text{eqn 19}$$

The stability criteria for the two models are compared in Fig. 7: the two models have complementary properties. In both models population cycles always occur when the parasite is able to establish, diverging cycles occur when the intrinsic equilibrium density of the grouse exceeds the threshold for establishment by a factor of about 4–6. Although short periods of larval arrestment (2–3 months) produce oscillations in Model II, longer periods of arrestment lead to damped cycles and eventually to asymptotically stable parasite host dynamics. While increased survival of the free-living stages always increases the propensity of the system to oscillate, increased larval arrestment has the opposite effect. Larval arrestment would seem to provide an interesting example of a time delay stabilizing a two species interaction!

FACTORS DETERMINING THE PERIOD OF HOST–PARASITE CYCLES

Standard analytical techniques may be employed to determine the length of the cycles observed in the model (Appendix 1). In Model I the period of the cycles is primarily determined by the life expectancy of the parasite larval stages and the intrinsic growth rate of the grouse (Fig. 7a). For the range of parameters given in Table 2, Model I tends to show cycles in host and parasite abundance that are

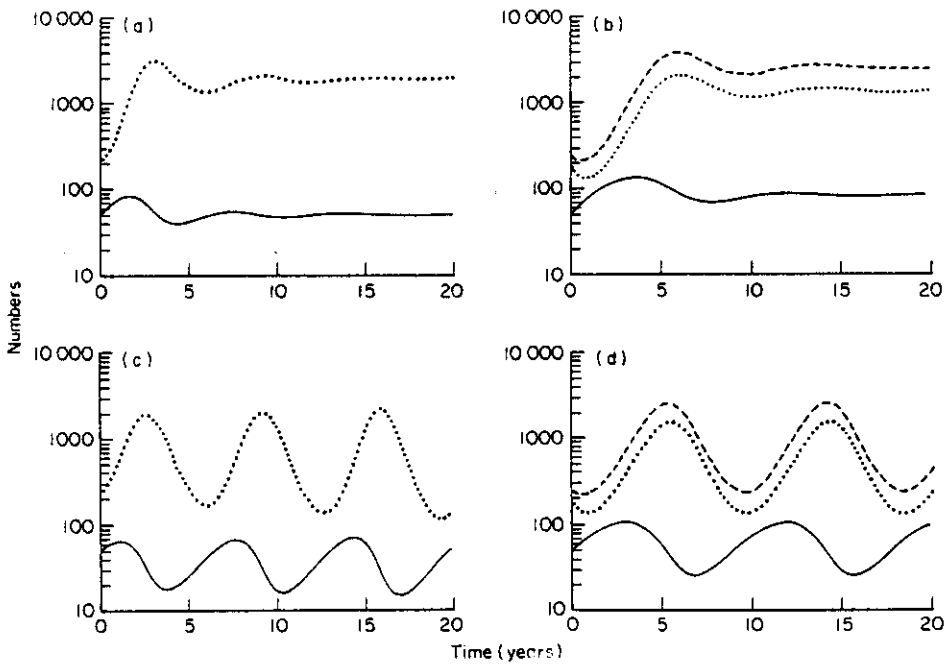

Fig. 6. A comparison of the effects of larval arrestment and parasite-induced reduction in host fecundity. All the results are depicted for a host population regulated by territoriality. In all cases the host populations and mean worm burdens are labelled as in Fig. 5. The parameter values are the same as those given in Figs 4 & 5 with $\delta = 0{\cdot}0005$. In the top figures (a,b) the parasite has no effect on host fecundity, the figures on the left (a,c) correspond to a parasite with no arrested stages (Model I), the figures on the right (b,d) depict the case where parasites arrest for 3 months (Model II).

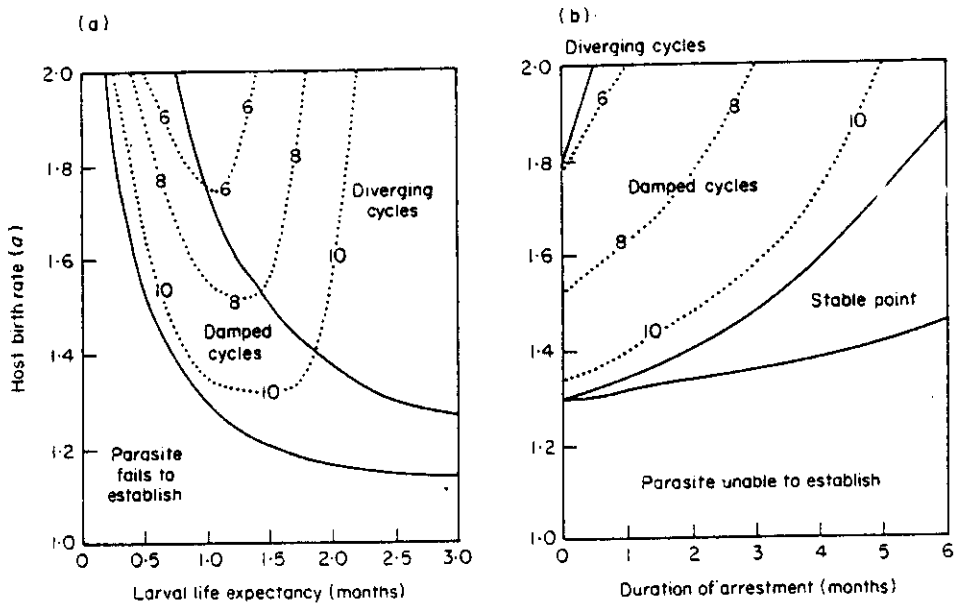

Fig. 7. The effects of variations in host fecundity and larval dynamics on the behaviour of populations of *T. tenuis* in grouse. In (a) we assume no arrestment, while in (b) we assume constant free-living larval life expectancy of 4 weeks ($\gamma = 13$). The solid lines delineate different regions of population behaviour, the broken contour lines give the expected cycle lengths in regions where cycles occur. All other parameter values are set to the mean values given in Table 2.

similar in period to those recorded by Potts, Tapper & Hudson (1984) for moors in the North of England. In contrast, Model II produces cycles with a time period of 7–10 years for a broad range of values of a and θ (Fig. 7b). These are more consistent with the cycles observed throughout Scotland (Mackenzie 1952; Williams 1985; Hudson & Dobson 1990). In both models, increases in the intrinsic growth rate of the host population lead to decreases in cycle length. Decreases in the duration of larval arrestment (Model II), or in the life expectancy of free-living stages of the parasite (Model I), increase the period with which the host–parasite system oscillates. These differences may account for the regional variation in cycle length recorded from grouse populations in different geographic locations (Hudson & Dobson 1990).

COMPARISON WITH EMPIRICAL DATA

The dynamics of the model that incorporates free-living larvae (Model I) more closely matches the dynamics of the population at the Gunnerside study site (compare Fig. 8 with Fig. 6c). The structure of the cycles in grouse abundance produced by the model correspond to those observed at Gunnerside with a slow period of host population growth followed by a period of rapid decline. During the initial period of population growth, mean parasite burdens initially decline, then increase as host population size peaks; note that the parasite decline does not start until the host population crash is well advanced. The cycle period of 5 years also corresponds closely to that observed at Gunnerside.

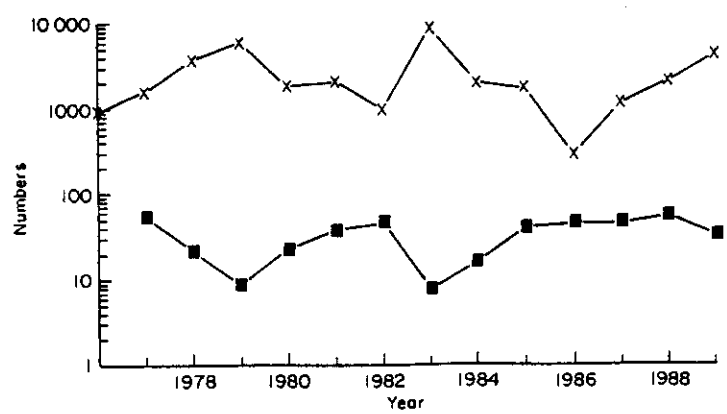

Fig. 8. Changes in numbers of grouse (hens breeding per km^2) (■) and mean parasite burden per grouse (×) over 14 years at Gunnerside. The methods used to collect the original data are described in Hudson, Newborn & Dobson (1992).

8

PROPORTIONAL ARRESTMENT

Evidence from a number of studies suggests that only a proportion of larvae enter hypobiosis (Schad 1977; Gibbs 1986; Shaw, Moss & Pike 1989). The dynamic consequences of proportional arrestment may be examined by modifying equations 3 and 5 to produce a hybrid model (Model III) that allows a fixed proportion of larvae to arrest. The numbers of grouse and free-living larvae are given by equations 1 and 4, changes in the numbers of arrested and adult parasites are now described by the following equations

$$dA/dt = \sigma\beta WH - (\mu_A + b + \theta)\,A - \alpha\frac{PA}{H} \qquad \text{eqn 20}$$

$$dP/dt = \theta A + (1-\sigma)\beta WH - (\mu_P + b + \alpha)P - \alpha\frac{P^2}{H}\left(\frac{k+1}{k}\right) \qquad \text{eqn 21}$$

Here σ is the proportion of larvae that enter an arrested stage. Expressions for both R_0 and H_T may be readily obtained for this hybrid model:

$$R_0 = \frac{T_1}{M_1 M_2}\left[(1-\sigma) + \frac{\sigma\theta}{M_3}\right] \qquad \text{eqn 22}$$

and

$$H_T = \frac{\gamma M_1}{\beta\left(\lambda\left[(1-\sigma) + \frac{\theta\sigma}{M_3}\right] - M_1\right)} \qquad \text{eqn 23}$$

These expressions suggest that the behaviour of this more complete model is intermediate to that of Models I and II. Note that when the proportion of larvae arresting equals either zero or unity, the original expressions for R_0 and H_T are obtained (equations 6–9).

The dynamic consequences of allowing a different proportion of larvae to arrest are illustrated in Fig. 9. As the proportion of larvae that enter a period of arrested development increases, the host and parasite populations exhibit more heavily damped cycles of longer frequency. Preliminary studies from the north of England suggest that a lower proportion of larvae enter arrestment than in the north-east of Scotland (Hudson, Newborn & Dobson, unpublished). This suggests that variation in the proportion of larvae arresting may also be important in determining differences in cycle period between grouse populations in these areas.

Discussion

The models developed by Anderson & May (1978) and May & Anderson (1978) predicted that under certain conditions, parasitic helminths could cause cyclic fluctuations in the abundance of their host populations. The application of these models to the empirical and experimental data collected on the grouse–*T. tenuis* system suggests that the main interaction causing the system to cycle is that the impact of the parasite on host fecundity is greater than its impact on host mortality. The relatively low levels of parasite aggregation observed in the grouse–*T. tenuis* system further increases the tendency of the system to oscillate. The model strongly suggests that the parasite is driving the cycles in grouse abundance observed in the long-term study at Gunnerside (Hudson, Newborn & Dobson 1992).

An interesting point to emerge from the simulations (Figs 5, 6 & 9) and the longitudinal study (Fig. 8) is that mean parasite burdens do not peak until after host population density has started to decline. When combined with the sublethal effects of the parasite on breeding production, this result may explain why previous workers (e.g. Jenkins, Watson & Miller 1963, 1967; Wynne-Edwards 1986) have concluded that the parasites were a consequence rather than a cause of population cycles. Moss and Watson (1985) have proposed

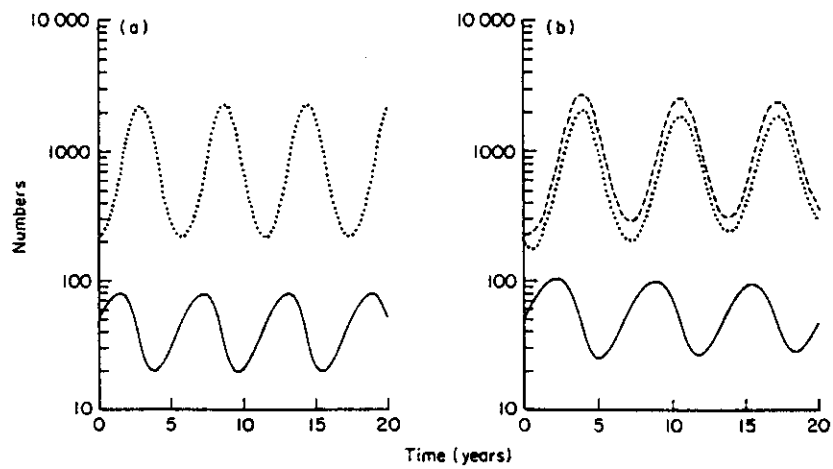

Fig. 9. The effects of proportional arrestment on the dynamics of Model III: (a) 5% of the larvae enter a period of arrestment after infecting the host; (b) 50% enter a period of arrestment. All the parameter values are the same as in Fig. 6b.

that cycles are 'adaptive irruptions' generated by 'intrinsic factors that operate through changes in spacing behaviour', which have 'evolved to avoid outbreaks of trichostrongylid worms'. It is difficult to see how adaptive irruptions could have evolved without invoking group selection (Wynne-Edwards 1986). A more recent suggestion is that cycles could be generated through differential aggression between kin and non-kin coupled with inversely density-dependent breeding success (Mountford *et al.* 1990; Moss & Watson 1991). However, simulations of this model demonstrate that the parameter values necessary to generate cycles are not consistent with field measurements of levels of deference towards kin.

The study described here suggests that cyclic fluctuations in grouse density may be generated by their interactions with parasites for wide ranges of parameter values. Variations in host population size and parasite burdens at different times in the cycle may produce variations in levels of aggression and dispersal that suggest these covariates are driving the cycles (Moss, Watson & Rothery 1984; Moss & Watson 1985). This is not to say that parasites cause all population cycles, but failure to differentiate cause from effect in earlier studies may be at the root of the controversies surrounding discussions of tetraonid cycles.

ANNUAL AND SEASONAL DYNAMICS

Empirical studies of *T. tenuis* reveal pronounced seasonal variation in both the survival of the parasite's free-living stages and their propensity to arrest (Watson 1988; Shaw 1988; Shaw, Moss & Pike 1989; Hudson & Dobson 1990; Hudson, Newborn & Dobson, 1992). The survival of free-living larvae is dependent on humidity and ambient temperature, while propensity to arrest is likely to be most pronounced as temperature variations become more severe both seasonally and geographically (Schad 1977; Gibbs 1986). The models discussed in this paper assume these rates remain constant. In reality, Model I corresponds to the situation operating in the spring and summer months while Model II corresponds to the autumn and winter months. A full description of the more complex seasonal models that include seasonal variation in the parasite's vital rates will be published subsequently (Dobson & Hudson, in press). Although this more complex model has a richer array of dynamic behaviour (particularly on time-scales operating within each calendar year), the main features of the analyses described above are not significantly altered by the inclusion of this seasonal forcing. Essentially, the longer-term dynamics are dominated by the parasites' density-dependent impact on host fecundity and the propensity of larvae to arrest their development. These two factors interact to produce the population cycles characteristic of regulated systems which incorporate time delays (May 1973; MacDonald 1978; Nunney 1985a,b).

This study suggests that interactions between red grouse and the parasitic nematode *T. tenuis* provide sufficient conditions for cycles to occur in grouse abundance. The definitive falsification of this hypothesis is to stop a population from cycling by controlling or eradicating parasites. This experiment is being undertaken at a variety of locations and initial results are encouraging (Hudson & Dobson 1990). All previous hypotheses that seek to explain cycles in grouse abundance have been falsified when rigorously tested (Watson, Moss & Parr 1984; Bergerud 1987; Watson *et al.* 1988; Hudson & Dobson 1990). The results from this study imply that the presence of *T. tenuis* is the primary factor causing population cycles of grouse in the North of England. The ubiquitous presence of the parasite in other areas at similar high intensities (Hudson & Dobson 1990; Wilson 1983) suggest that it may be a significant factor in a number of areas in Scotland. An increased propensity for the larvae to arrest in the North of Scotland may cause the longer cycles observed in this area.

The results described here encourage examination of the potential role of parasites in other species which illustrate long-term cycles in abundance. In particular, we caution that the subtle roles played by hypobiotic larvae and the parasites' sublethal impact on host fecundity may have caused parasites to have been overlooked in previous studies seeking to determine why animal populations exhibit cyclic fluctuations.

Conclusions

Interactions with the parasitic nematode *T. tenuis* generate population cycles in red grouse through their time-delayed impact on fecundity. Variations in cycle period between areas can be explained through changes in the propensity of larvae to arrest and changes in the growth rate of the grouse population. The period of the cycles varies directly with the duration of larval arrestment and inversely with host birth rates. When the life expectancy of the parasite free-living larvae is low, transmission of the parasite will be sporadic and grouse numbers will not cycle regularly. Under these conditions grouse density will be regulated by territorial behaviour and its interactions with other natural enemies, such as predators (Hudson & Dobson 1992). Although the territorial 'spacing behaviour' of the grouse stabilizes the more extreme dynamical effects of parasite arrestment, it does not generate population cycles in grouse.

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Appendix 1

This appendix outlines the formal stability analysis of Models I & II. The models may be simplified by grouping together sets of related parameters; here we set $c_1 = a - b$, $c_2 = \alpha + \delta$, $c_3 = \mu_P + b + \alpha$, $c_4 = \alpha (k+1)/k$ and $c_5 = \mu_A + b + \Theta$. Noting that $d' = c_3 + c_1 c_4/c_2$, we can consider the fate of a small perturbation about the equilibrium, H^*, W^*, P^*.

Model I

Writing $H_{(t)} = H^* + x_{(t)}$, $W_{(t)} = W^* + y_{(t)}$ and $P_{(t)} = P^* + z_{(t)}$, and linearizing by neglecting higher order terms, we obtain for model I (equations 1–3):

$$dx/dt = c_1 x - c_2 z \qquad \text{eqn A1}$$

$$dy/dt = -d' M^* x - \gamma\left(\frac{\lambda}{\lambda - d'}\right) + \lambda z \qquad \text{eqn A2}$$

$$dz/dt = M^*(M^* c_4 + d') + \frac{d'\gamma}{\lambda - d'}\, y - (d' + M^* c_4) z \qquad \text{eqn A3}$$

The temporal behaviour of $x_{(t)}$, $y_{(t)}$ and $z_{(t)}$ then goes as $\exp(\lambda t)$, where the damping rates are given from equations A1–A3 by the quadratic equation

$$\Lambda^3 + A\Lambda^2 + \Lambda B + C = 0 \qquad \text{eqn A4}$$

I Model I, A, B and C are given by

$$A \equiv \gamma\left(\frac{\lambda}{\lambda - d'}\right) + d' + (a - b)\left(\frac{\alpha k}{\alpha + \delta} - 1\right) \qquad \text{eqn A5}$$

$$B \equiv \gamma\left(\frac{\lambda}{\lambda - d'}\right)(a - b)\left(\frac{\alpha k'}{\alpha + \delta} - 1\right) \qquad \text{eqn A6}$$

$$C \equiv \gamma d'(a - b) \qquad \text{eqn A7}$$

The Routh-Hurvitz criteria for local stability are that A, B and C all be greater than zero and that $AB > C$ (May 1973). A and B will only be greater than zero when $\alpha/k > \delta$; this requires either k to be small or $\alpha > \delta$. These conditions are rarely met in the grouse–*T. tenuis* system. The further criteria $AB > C$ is only met when

$$\left(\frac{\lambda}{\lambda - d'}\right)\left(\frac{\alpha/k - \delta}{\alpha + \delta}\right)\left[\gamma\left(\frac{\lambda}{\lambda - d'}\right)\right] > d' \qquad \text{eqn A8}$$

when $\lambda \gg d'$, this simplifies to

$$\gamma > d' k\left(\frac{\alpha + \delta}{\alpha - \delta k}\right) \qquad \text{eqn A9}$$

Model II

The stability analysis for Model II follows similar lines, the stability-determining damping rates are now determined by the following expressions for A, B and C:

$$A \equiv d' + d'' + (a - b)\left(\frac{\alpha k'}{\alpha + \delta} - 1\right) \qquad \text{eqn A9}$$

$$B \equiv d'd'' + (a - b)\left(d''\left(\frac{\alpha k'}{\alpha + \delta} - 1\right) - d'\left(\frac{\delta}{\alpha + \delta}\right)\right) - \frac{\theta H^* \lambda}{H^* + H_0} \qquad \text{eqn A10}$$

$$C \equiv (a - b)\left(\frac{\theta H^* \lambda}{(H^* + H_0)}\left(\frac{H^* + 2H_0}{(H^* + H_0)}\right) - d'd''\right) \qquad \text{eqn A11}$$

The Routh-Hurvitz stability criteria still require $\alpha/\delta > k$, for $A > 0$. More complex conditions now determine the sign of the second inequality; because $d'd'' \simeq \Theta H^* \lambda/(H^* + H_0)$, B may be simplified to $\alpha/\delta > k(d'/d'' + 1)$. This requires high levels of aggregation ($k \ll 1$), or short periods of larval arrestment ($\Theta > 1$), the former condition is rarely found in the grouse–*T. tenuis* system. The same substitution suggests that $C > 0$ for all positive H^*. Although the requirement that $AB > C$ is not easily simplified, extensive numerical explorations suggest it is only satisfied when H^* is small.

Periodicity of the cycles

When the inequalities described in this appendix are transgressed the system has a propensity to oscillate. The period of these cycles will scale with $2\pi/w$, where $w = (s1 - s2)\sqrt{3}/2$. Here $s1 = [r + (q^3 + r^2)^{1/2}]^{1/3}$, and $s^2 = [r - (q^3 + r^2)^{1/2}]^{1/3}$, with $q = 0{\cdot}33B - 0{\cdot}11A^2$, and $r = 0{\cdot}17(AB - 3C) - 0{\cdot}037A^3$. Figures 7 and 9 in the main text illustrate the influence of host birth rate and duration of larval arrestment on cycle length.